Remarks

Reconsideration and withdrawal of the rejections of the claims, in view of the amendments and remarks herein, is respectfully requested. Claims 1, 10 and 87-89 are amended, and claim 5 is canceled. The amendments are intended to further prosecution and are not intended to concede to the correctness of the Examiner's position or to prejudice the prosecution of the claims prior to amendment, which claims are present in an application related to the present application. Claims 1-4, 6-12, 29-84, and 86-89 are pending.

The Examiner is respectfully thanked for the courtesies extended to Applicant's Representative in the telephonic interview conducted on April 14, 2005, in which the rejections of the claims were discussed.

The Examiner rejected claims 1-12, 29-36, 83-84, 86, and 88 under 35 U.S.C. § 112, first paragraph, as failing to comply with the written description requirement. The amendments to claims 1 and 88 obviate this rejection.

The Examiner also rejected claims 88 and 89 under 35 U.S.C. § 112, first paragraph, as failing to comply with the written description requirement (a "new matter" rejection). Specifically, the Examiner asserts that there is no explicit support in the specification for identifying an agent that enhances "internalized adeno-associated virus" transport to the nucleus. This rejection is respectfully traversed.

The Examiner is requested to consider that the specification discloses that agents within the scope of the invention include those which enhance virus transduction by enhancing viral endocytosis, decreasing viral nucleic acid or protein degradation in endosomes, and/or enhancing viral transport to the nucleus (page 4, lines 29-31) and that virus binding and endocytosis of adeno-associated virus (AAV) at the apical membrane is not the major rate limiting step for transduction of airway epithelial cells (page 5, lines 1-4). Rather, it is disclosed that endosomal processing and trafficking of internalized virus to the nucleus is a limiting step for AAV transduction (page 5, lines 5-7; emphasis added) and that proteosome inhibitors such as LLnL may increase endosomal processing and routing of AAV to the nucleus (see Figure 15 and page 77, line 6).

In Example 3, labeled AAV was bound to cells at 4°C and after a shift to 37°C, the location of the labeled virus in cells was detected. After 1 hour at 37°C, it is disclosed that virus was observed in the cytoplasm of infected cells ("internalized" virus) and by 2 hours, virus was observed in the nucleus of infected cells. In Example 6, it is disclosed that the extent of viral internalization was detected. After binding virus to cells for 1 hour at 4°C and then increasing the temperature to 37°C, the cells were treated with trypsin and washed to remove any remaining extracellular virus. The first time point for which data was collected after raising the temperature to 37°C was 4 hours, 2 hours after virus can be observed in the nucleus (see Example 3 and page 71, line 20). Note that it is disclosed that viral binding and internalization were not affected by LLnL treatment (page 77, line 11).

Accordingly, withdrawal of the § 112(1) "new matter" rejection is appropriate and is respectfully requested.

The Examiner rejected claims 1, 4-12, 29-36, and 88 under 35 U.S.C. § 102(b) as being anticipated by Nair et al. (U.S. Patent No. 5,831,068). This rejection, as it may be maintained with respect to the pending claims, is respectfully traversed.

Nair et al. disclose a method in which a cell is contacted with an antigenic peptide, resulting in a potent antigen presenting cell which can be administered to a mammal to treat or prevent cancer or infection (abstract). The method includes inhibiting the activity of a MHC class I pathway-associated component (e.g., TAP or a proteosome) prior to contact with an antigen (column 1, lines 60-67). It is disclosed that inhibition of the activity of a MHC class I pathway-associated component may be accomplished with a proteosome inhibitor such as LLnL (column 2, lines 30-33). The inhibition of the function of one or more components of the class I antigen processing pathway results in cells deficient in endogenous peptide loading (column 2, lines 43-45). When those cells are contacted with exogenous antigenic peptide, empty class I molecules are loaded with that peptide (column 2, lines 45-50).

The only mention of viruses in Nair et al. is in the context of the use of viral-specific antigenic peptides to prepare viral antigen presenting cells for treating or preventing viral infection (abstract, column 3, lines 25-27, column 4, lines 1-3, column 20, lines 46-49 and 56-61, and column 22, lines 5-10).

Nair et al. do not teach a method to identify agents that enhance AAV transduction in mammalian cells contacted with an agent and AAV. Therefore, withdrawal of the § 102(b) rejection is respectfully requested.

Conclusion

Applicant respectfully submits that the claims are in condition for allowance, and notification to that effect is earnestly requested. The Examiner is invited to telephone Applicant's attorney at (612) 373-6959 to facilitate prosecution of this application.

If necessary, please charge any additional fees or credit overpayment to Deposit Account No. 19-0743.

Respectfully submitted,

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Date May 18, 2005 By _____
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Reg. No. 39,665

CERTIFICATE UNDER 37 CFR 1.8: The undersigned hereby certifies that this correspondence is being deposited with the United States Postal Service with sufficient postage as first class mail, in an envelope addressed to: MS Amendment, Commissioner of Patents, P.O. Box 1450, Alexandria, VA 22313-1450, on this 18 day of May, 2005.

CANDIS BUENDING _____

Name Signature